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How to? Nasıl yapalım?

Reduced biventricular pacing: What is the mechanism and how to manage it?

Azalmış biventriküler pacing: Mekanizma nedir ve nasıl yaklaşalım?

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Case presentation

A 43-year-old male with a history of dilated cardiomyopathy and a cardiac resynchronization therapy (CRT) defibrillator (Unify Quadra; St. Jude Medical Inc., St. Paul, MN, USA) implanted 5 years earlier for primary prevention presented at the cardiac rhythm and device clinic for regular follow-up. Device interrogation showed normal parameters (Table 1). The device was programmed in DDD mode at 60 ppm with sensed and paced atrioventricular (AV) delays of 120 milliseconds and 150 milliseconds, respectively. Biventricular pacing was at 50%. The patient's clinical and echocardiographic situation had not improved since CRT implantation. The echocardiogram revealed a severely dilated left ventricle with an ejection fraction of 20%. The device diagnostic summary showed 48% premature ventricular complexes (PVCs), inhibiting biventricular pacing (Fig. 1). However, there were no PVCs observed during device interrogation. What are the next steps? What caused the reduced biventricular pacing, and what circumstances should be considered in this case?

Discussion

A 24-hour Holter cardiac monitor device was requested to assess the correlation between the rhythm tracings and device diagnostics, and to determine the morphology and burden of possible PVCs. The Holter monitor report indicated that there were occasional PVCs with frequent episodes of non-paced ventricular rhythms without preceding atrial activity (Fig. 2). The following reasons and potential solutions for reduced biventricular pacing were considered:

Abbreviations:

AV	*Atrioventricular*
CRT	*Cardiac resynchronization therapy*
PVARP	*Postventricular atrial refractory period*
PVC	*Premature ventricular complex*

1. Atrial lead dislodgement: Dislodgement of a lead can prevent the CRT from tracking the initial atrial rhythm, thereby causing AV dyssynchrony.[1] In this case, both atrial pacing in AAI mode and chest radiography showed intact leads, which ruled out this diagnosis.

2. Atrial fibrillation with conducted ventricular activity: Device interrogation and 24-hour rhythm Holter results ruled out this diagnosis.

3. T-wave oversensing: T-wave oversensing is a common cause of inappropriate implantable cardioverter-defibrillator therapies and reduced biventricular pacing.[2] In this case, T-wave oversensing was not detected during device interrogation.

4. PVC-induced locking of the P-waves in the postventricular atrial refractory period (PVARP). During sinus rhythm and biventricular pacing, PVCs or T-wave oversensing can initiate the PVARP and shift the pacemaker timing so that the subsequent P-wave falls within the PVARP and conducts to the ventricle, causing a spontaneously sensed QRS complex. Junctional rhythm can also push the P-waves into the PVARP and

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Table 1. Lead parameters

	Sensing, mV	Threshold, V/ms	Impedance, Ohm	Pacing, %
Right ventricle	2.1	0.5/0.40	410	22
Right atrium	11.4	0.5/0.40	360/45	50
Left ventricle	NA	0.5/0.40	900	50

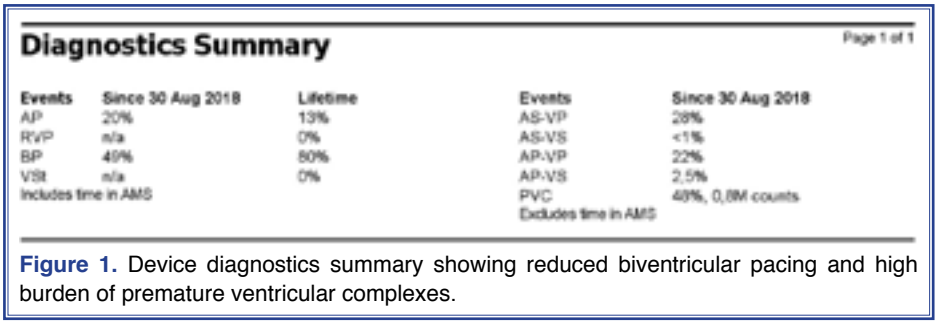

Figure 1. Device diagnostics summary showing reduced biventricular pacing and high burden of premature ventricular complexes.

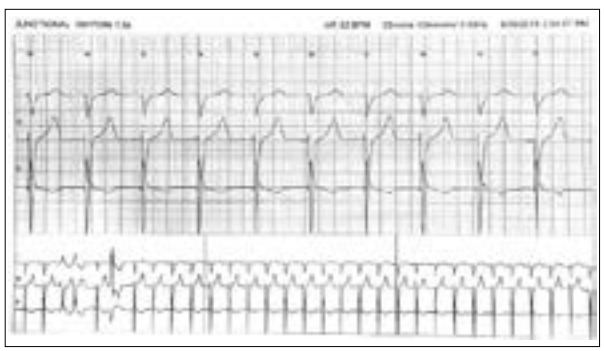

Figure 2. A 24-hour Holter recording showing accelerated junctional rhythm at 80 bpm with retrograde P-waves.

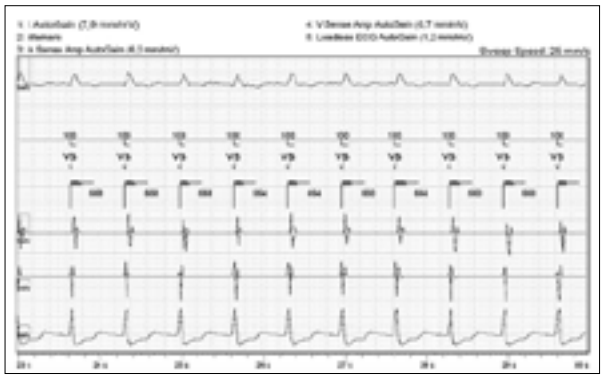

Figure 3. Device recording depicting simultaneous atrial and ventricular electrograms and atrial activities falling in the post ventricular blanking period. Note that there is no visible atrial activity (P-wave) in the electrocardiogram channel.

promote the intrinsic rhythm. This was confirmed with Holter monitoring (Fig. 2) and with the intracardiac electrograms stored in the device (Fig. 3). Both tracings depicted simultaneous ventricular and atrial activities and supported the diagnosis of an accelerated junctional rhythm at 80 bpm. In this case, we added a beta-blocker (Bisoprolol 5 mg once daily by mouth) in order to suppress AV node automaticity. One month after beta-blocker initiation, follow-up biventricular pacing had increased to 88%. Other possible solutions could be to increase the lower rate or catheter ablation in case of failure to respond to medication.

This case highlights the importance of recognizing misinterpretation of the device diagnosis, which could lead to errors in medical decisions. In this case, the reason for suboptimal biventricular pacing was solved with basic electrocardiographic interpretation.

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